CHAPTER 47
Disorders of the Pericardium

KEY TEACHING POINTS

- Acute pericarditis is principally defined by bedside criteria: combinations of characteristic chest pain, pericardial rub, and electrocardiographic changes. The pericardial rub is *less* frequent in neoplastic pericarditis compared with other etiologies.
- The diagnosis of cardiac tamponade combines bedside criteria—elevated neck veins, tachycardia, pulsus paradoxus—with echocardiographic criteria. In patients with pericardial effusions, a pulsus paradoxus greater than 12 mm Hg accurately identifies those patients whose cardiac output improves after pericardiocentesis.
- The key physical findings of constrictive pericarditis are elevated neck veins, prominent *y* descent in venous waveforms, pericardial knock, and hepatomegaly.

PERICARDITIS AND THE PERICARDIAL RUB

I. INTRODUCTION

The pericardial rub is a physical sign of pericarditis, or inflammation of the pericardium, which is caused by a wide variety of disorders, including infections, connective tissue diseases, radiation, myocardial infarction, neoplasia, uremia, and trauma.

In the 1820s, shortly after the introduction of the stethoscope, Collin first described the pericardial rub as a sound "similar to that of the crackling of new leather."[1]

II. THE FINDING

Pericardial rubs are grating, scratching, or creaking sounds that are loudest near the left sternal border and are most apparent when the patient is sitting upright, leaning forward, and holding his or her breath in deep expiration.[2,3] They resemble the sound of two pieces of sandpaper being rubbed together. Compared with heart murmurs, the pericardial rub has more high-frequency energy and sounds closer to the ear;[2] it may completely disappear during inspiration or expiration, and up to one-fourth are palpable.[3,4]

In approximately 50% of patients, the rub has three components per cardiac cycle—one during ventricular systole and two during diastole (mid-diastole and atrial systole).* In approximately one-third of patients, only two components are heard (usually the atrial and ventricular systolic rub), and in the remaining 15%, only a single-component ventricular systolic rub is heard.[3]

*These three components represent the three moments in the cardiac cycle when the ventricle is moving the most.

III. CLINICAL SIGNIFICANCE

A. THE RUB AND PERICARDITIS

Because the diagnosis of pericarditis relies on bedside criteria, one of which is the rub, the diagnostic accuracy of the rub cannot be assessed. The other two bedside criteria for pericarditis are the characteristic pericardial chest pain (precordial pleuritic pain radiating to the trapezius ridge that is relieved sitting up) and the characteristic electrocardiographic changes (diffuse concave ST elevation, PR segment depression, absence of Q waves).[5,6] Most clinical studies of pericarditis require two of these three criteria. Only 50% to 66% of patients with pericarditis have detectable pericardial effusions on echocardiography.[5-7]

B. THE RUB AND PERICARDIAL EFFUSION

Although the pericardial rub suggests the rubbing together of contiguous pericardial surfaces, the sound often persists after accumulation of significant pericardial effusions.[3,8] The rub is heard, for example, in up to one-fourth of patients with cardiac tamponade (see later). Therefore the *presence* of the rub cannot be used to argue *against* the development of pericardial effusion.

C. THE RUB AND NEOPLASTIC DISEASE

Pericardial rubs are less frequent in neoplastic pericarditis compared with other etiologies. For example, in patients with known cancer who subsequently develop pericardial disease, the presence of a rub increases the probability that the pericarditis is idiopathic or radiation-induced, not neoplastic pericarditis (positive likelihood ratio [LR] = 5.5, negative LR = 0.4).[9] In another study of 322 patients presenting with undiagnosed moderate or severe pericardial effusion, the presence of a pericardial rub (among other inflammatory signs[†]) increased the probability of a nonneoplastic etiology (LR = 2.3).[10]

D. THE RUB AND MYOCARDIAL INFARCTION

A pericardial rub is found in 5% to 20% of patients with acute myocardial infarction, usually appearing between hospital days 1 and 3.[11-15] The incidence is lowest (i.e., 5% to 7%) in patients receiving immediate thrombolytic medications or angioplasty.[13,15] Compared with patients who do not develop rubs, patients with rubs have significantly larger myocardial infarctions, lower ejection fractions, more extensive coronary artery disease, and more complications, including congestive heart failure and atrial arrhythmias.[11,13,14] In these patients, however, tamponade is rare, even if they receive thrombolytic medications.[13]

CARDIAC TAMPONADE

I. INTRODUCTION

Cardiac tamponade occurs when a pericardial effusion has become so large and tense that intrapericardial pressures exceed normal filling (i.e., diastolic) pressures of the heart, thus impairing diastolic filling of the heart and reducing cardiac output.

[†] In this study, inflammatory signs were defined as two or more of the following: pericardial rub, characteristic pericarditis chest pain, fever, or characteristic ECG changes.

The history of diagnosing tamponade illustrates well the tension that sometimes exists between older diagnostic standards based on physical signs and newer ones based on clinical imaging. For example, early descriptions of tamponade, which were based on catastrophic acute intrapericardial hemorrhage, emphasized hypotension, elevated neck veins, and the small, quiet heart as diagnostic findings **(Beck triad)**.[16,17] Later, after it became obvious that many medical patients with tamponade had normal blood pressure and loud heart tones, the definition of tamponade shifted to emphasize large pericardial effusions, elevated neck veins, pulsus paradoxus, and relief of symptoms and signs after pericardiocentesis.[18] Finally, in the 1980s several echocardiographic criteria for tamponade were introduced,[17,19] although studies have subsequently shown that relying solely on echocardiographic criteria sometimes identifies patients who fail to improve symptomatically or physiologically after pericardiocentesis.[20-22]

Therefore the diagnosis of tamponade should not rely solely on the echocardiographic report but requires synthesis of all the findings, emphasizing especially the ones from physical diagnosis—elevated neck veins, tachycardia, and pulsus paradoxus.[23]

II. THE FINDINGS

Table 47.1 presents the physical signs observed in several studies of patients with proven cardiac tamponade; most patients present with shortness of breath.[18,27] The

TABLE 47.1 Cardiac Tamponade*

Physical Finding†	Frequency (%)‡
NECK VEINS	
Elevated neck veins	100
Kussmaul sign	0
ARTERIAL PULSE	
Tachycardia (>100 beats/min)	81-100
BLOOD PRESSURE	
Systolic blood pressure greater than 100 mm Hg	58-100
Pulsus paradoxus >10 mm Hg	98
Pulsus paradoxus >20 mm Hg	78
Pulsus paradoxus >30 mm Hg	49
Pulsus paradoxus >40 mm Hg	38
Total paradox	23
AUSCULTATION OF HEART	
Diminished heart tones	36-84
Pericardial rub	27
OTHER	
Hepatomegaly	58
Edema	27

*Diagnostic standard: for *tamponade,* cardiac output that improved after drainage of pericardial effusion.
†Definition of finding: for *total paradox,* palpable pulse disappears completely during inspiration.
‡Results are overall mean frequency or, if statistically heterogeneous, the range of values.
Data from 121 patients based upon references 18 and 24-27.

TABLE 47.2 Constrictive Pericarditis*

Physical Finding	Frequency (%)†
NECK VEINS	
Elevated neck veins	95
Prominent y descent (Friedreich sign)	57-100
Kussmaul sign	21-50
ARTERIAL PULSE	
Irregularly irregular (atrial fibrillation)	36-70
BLOOD PRESSURE	
Pulsus paradoxus >10 mm Hg	17-43
AUSCULTATION OF HEART	
Pericardial knock	28-94
Pericardial rub	3-16
OTHER	
Hepatomegaly	53-100
Edema	70
Ascites	37-89

*Diagnostic standard: for *constrictive pericarditis*, surgical and postmortem findings,[24,29,30,33,37] sometimes in combination with hemodynamic findings.[31,32,34-36]
†Results are overall mean frequency or, if statistically heterogeneous, the range of values.
Data from 282 patients based upon references 24 and 29-37.

definition and pathogenesis of pulsus paradoxus and elevated neck veins are discussed in Chapters 15 and 36.

The three key findings of tamponade are elevated neck veins (100% of patients), tachycardia (81% to 100% of patients), and pulsus paradoxus greater than 10 mm Hg (98% of patients). In patients with pericardial effusions the finding of pulsus paradoxus greater than 12 mm Hg detects tamponade with a sensitivity of 98%, specificity of 83%, positive LR of 5.9, and negative LR of 0.03 (see Chapter 15).[28]

Cardiac tamponade is one of the few causes of elevated neck veins with an absent y descent (see Chapter 36). This contrasts sharply with the exaggerated y descent of constrictive pericarditis (see later).

CONSTRICTIVE PERICARDITIS

I. INTRODUCTION

Constrictive pericarditis is present when calcification or fibrosis of the pericardium impairs diastolic filling, thus causing elevated venous pressure and reduced cardiac output.

II. THE FINDINGS

Table 47.2 presents the physical signs of patients with constrictive pericarditis; most patients present with edema, abdominal swelling, and dyspnea.[33,34,38] The key

physical findings are elevated neck veins (95%), prominent *y* descent in venous waveform (57% to 100%, median 94%), pericardial knock (28% to 94%), and hepatomegaly (53% to 100%).

A. NECK VEINS

In addition to elevated venous pressure, the venous waveform displays an unusually prominent *y* descent, which combined with an exaggerated *x′* descent creates two conspicuous dips per cardiac cycle, making the waveform appear to trace an M or W with each arterial pulse (**Friedreich sign**, see Chapter 36). Sometimes these movements are transmitted to the liver, causing it to pulsate inward twice with each cardiac cycle.[39]

The prominent *y* descent occurs because diastolic filling is impaired only during the last two-thirds of diastole. At the moment the tricuspid valve opens (beginning of diastole and beginning of *y* descent), the right atrium empties rapidly and without resistance (causing a prominent *y* descent), although eventually the relaxing ventricle meets the limits of the rigid pericardial shell and pressures again increase.[40] This contrasts with tamponade, which impairs diastolic filling throughout diastole and thus eliminates the *y* descent.

B. KUSSMAUL SIGN

Kussmaul sign is the paradoxical increase in venous pressure during inspiration. This sign, present in 21% to 50% of patients with constriction, is discussed fully in Chapter 36 (an excellent video of Kussmaul sign is available in the article by Mansoor and Karlapudi).[41]

C. PERICARDIAL KNOCK

The pericardial knock is a loud, high-frequency early diastolic sound heard between the apex and left lower sternal border. It is discussed in Chapter 42.

D. OTHER FINDINGS

Up to 90% of patients with constrictive pericarditis have systolic retraction of the apical impulse (see Chapter 38).[35,42]

According to traditional teachings, pulsus paradoxus is a not a finding of constrictive pericarditis, yet the studies reviewed in Table 47.2 indicate that pulsus paradoxus does appear, occurring in 17% to 43% of patients with constrictive pericarditis.[24,29,34,38] This seeming contradiction probably reflects different definitions of pulsus paradoxus. When pulsus paradoxus is defined as 10 mm Hg or more inspiratory fall in systolic blood pressure (i.e., the usual definition), 17% to 43% of patients with constriction have the finding;[24,38] when it is instead defined as 20 mm Hg or more inspiratory fall, no patient has the finding.[24] In contrast, the usual pulsus paradoxus in patients with tamponade is 20 to 50 mm Hg (see Table 47.1).[18]

Therefore mild degrees of pulsus paradoxus (10 to 20 mm Hg) are commonly observed in patients with constrictive pericarditis, but larger degrees (>20 mm Hg) are not and suggest tamponade or another cause of the finding (see Chapter 15).

The references for this chapter can be found on www.expertconsult.com.

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